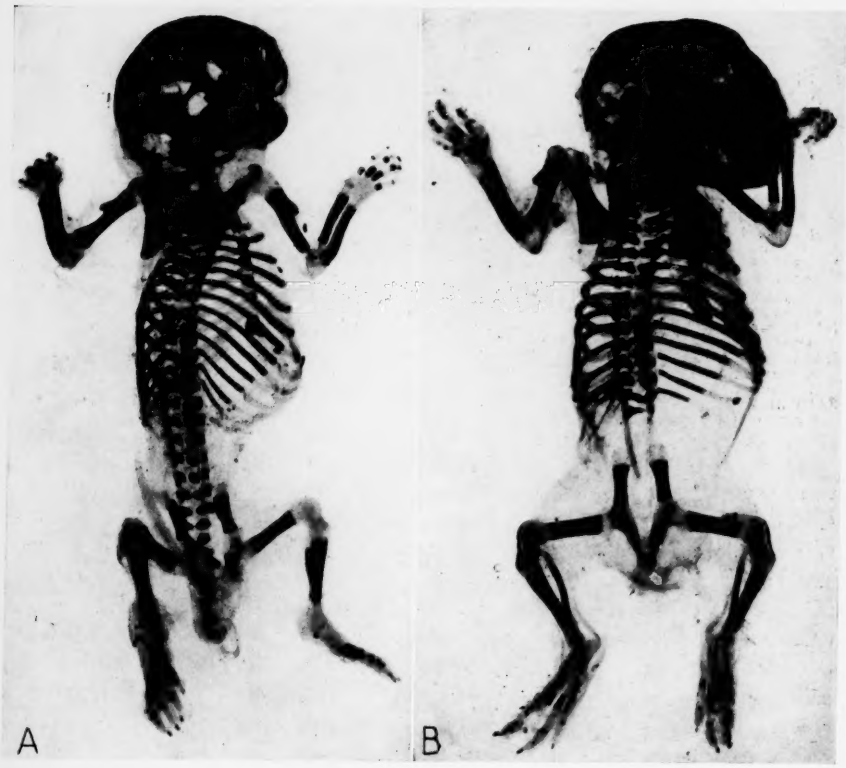
The Journal of HEREDITY



ONE GENE CAUSES A SHORT TAIL: TWO A SHORT SPINAL COLUMN

(SEE PAGE 343)

Hybrid Vigor in Maize Embryos

Associated Defects of Body and Mind

Plus and Minus Hybrid Vigor in Vetch Hybrids

Heredity and Congenital Malformations

A Color-Chimera in Sorghum

VOLUME 31 AUGUST, 1940 NUMBER 8

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The

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TENTH MEETING OF THE BIOLOGICAL PHOTOGRAPHIC ASSOCIATION

The tenth annual convention of the Biological Photographic Association will be held at the Hotel Schroeder, Milwaukee, Wisconsin, September 12, 13 and 14. Scientific photographers from all parts of the country will meet to exchange ideas and information on still and motion picture photography as well as the latest developments in color work. Formal papers will be presented outlining new methods, and there will be informal round-table discussions which will be especially instructive. A salon consisting of natural color and monochrome prints of biologic and clinical subjects will illustrate a very fine degree of perfection in biologic photography.

The membership of the Biological Photographic Association is composed of professional clinical and biologic photographers as well as physicians, dentists and scientists who are interested in this specialized branch of photography.

Further information concerning the convention program, salon specifications and membership in the society may be had by writing to the Secretary of the Biological Photographic Association, University Office, Magee Hospital, Pittsburgh, Pa.

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Photograph by Charles M. Rick

A CHROMOSOME-DEFICIENT POLLEN GRAIN

Frontispiece

The normal chromosome complement (*2n*) in *Tradescantia* is six pairs. The pollen grains (1*n*) contain normally six *un*paired chromosomes. In diploid *Tradescantia* unequal distribution of chromosomes at meiosis is not uncommon, but viable pollen grains lacking a chromosome (*n*-1) are extremely rare and microspores lacking two chromosomes were not found at all in diploid species. This photograph shows one of the few *n*-1 pollen grains found during this investigation.

CHROMOSOME DEFICIENCIES IN MICROSPORES OF *TRADESCANTIA**

ALAN D. CONGER
Biological Laboratories, Harvard University

MICROSPORES with less than the basic haploid complement of chromosomes are very rare in plants. Winge reported[10] microspores deficient for two chromosomes in *Vallisneria spiralis;* Belling found[1] an *n*-1 microspore in *Uvularia;* and Levan[5] found eight microspores deficient for one or more chromosomes in *Allium* hybrids. In diploid *Tradescantias* several deficient microspores have been found. In *Tradescantia,* and presumably in the other genera, microspores with duplicated chromosomes are relatively frequent.

If deficient microspores be fully viable, their frequency should be equal to that of microspores with additional chromosomes, since duplicate and deficient microspores are produced reciprocally by unequal segregation of chromosomes at meiosis. The fact that deficient microspores are comparatively rare indicates that the deficiencies are lethal, but the fact that they occur at all raises the question of how they survive to undergo the division of the microspore nucleus. These problems have been studied in a population derived from a natural hybrid between diploid *Tradescantia* species.

The frequency of aneuploid microspores varied in different plants, and variations were also associated with differences in weather or seasonal changes. This variation is to be expected since both genetic and environmental factors are known to affect chromosome pairing and segregation at meiosis. It was also found that anthers in which the microspores were just beginning nuclear division included fewer aneuploids than microspores which were nearing the end of the division cycle. In view of the various factors which may affect the frequency of production of aneuploid microspores, no detailed analysis was made of differences in individual plants, variation in environmental conditions, or effect of the time in nuclear cycle on the frequency of chromosome duplications and deficiencies.

The total frequencies of all the aneuploid and normal microspores examined, and the theoretical frequencies if all microspores survive, are shown in Table I. A relatively large number (4.2 per cent), of the microspores had more or fewer chromosomes than the basic genom. There were, however, only three deficient microspores, where 142 might have been expected if all had been viable.

The rare occurrence of deficient microspores is in striking contrast with the frequency of microspores with duplicated chromosomes. Since most of the aneuploid microspores result from unequal segregation at the first meiotic division, the deficient microspores must be produced as frequently as those with extra chromosomes. In fact, the deficient microspores might be expected to be more numerous because of the occasional loss of univalent chromosomes at meiosis.

The actual frequencies with which $n-1$ and $n+1$ microspores are produced at meiosis was determined from observations at anaphase of the first meiotic di-

TABLE I. Observed and theoretical frequencies of normal and aneuploid microspores. $n = 6$.

	$n-2$	$n-1$	n	$n+1$	$n+2$
Observed frequency	0	3	3333	138	4
Theoretical frequency	4	138	3333	138	4

*This paper is a part of a thesis submitted in partial fulfillment of the requirements for a B.S. degree at Harvard University.

vision. Two plants which had shown a high frequency of aneuploid microspores were used for an analysis of meiotic irregularities. The data are shown in Table II. In both plants there was a relatively high frequency of univalent formation and unequal segregation, about 8 per cent in plant 346, and 14.6 per cent in the "Harvard hybrid."

It is evident from the data in Tables I and II that duplicate and deficient microspores are formed in about equal numbers, but that few of the *n*—1 microspores survive. Deficient microspores in tetraploid plants can survive because of the duplication of genoms, and in this case the relative frequencies of aneuploid and normal microspores should represent the frequency of unequal chromosome segregation at meiosis. Observations on tetraploid species showed this to be true (Table III). Actually the deficient microspores are more frequent than those with extra chromosomes, as would be expected, since occasional univalents at meiosis fail to be included in either daughter nucleus.

Discussion

Although unequal segregation of homologous chromosomes occurs frequently at meiosis in the diploid *Tradescantia* plants examined, very few of the *n*—1 microspores so formed survive. In tetraploids, on the other hand, the deficient microspores are more frequent than those with extra chromosomes. The rare occurrence of *n*—1 microspores, — only two per cent of the expected number,—indicates that deficient microspores which survive to the microspore division are not produced by "non-disjunction" at the first meiotic division. How then are they produced?

In plants heterozygous for segmental interchange chromosomes, the microspores bearing a segmental deficiency produced at the first meiotic division undergo the second meiotic division unhindered, but after that time the deficiency prevents the following mitotic division, and such deficient microspores die.

It has been suggested by Sax and Edmonds[9] that the chromosomes at division pass off into the cytoplasm a "gene product" which conditions the cytoplasm for the next division. Thus, in this case the cytoplasm was completely conditioned at the first meiotic division, since all the chromosomes were present, but at the second meiotic division the cytoplasm of a deficient microspore would be incompletely conditioned and no further nuclear division would be possible. The fact that the frequency of sterile pollen grains has been found to be closely correlated with the frequency of "non-disjunctional" segregation of the ring or chain of four chromosomes in *Tradescantia* heterozygous for segmental interchange,[8] offers support for this theory.

The rare occurrence of *n*—1 microspores could be attributed to unequal segregation of chromosomes at the second meiotic division. Incomplete conditioning of the cytoplasm would not occur in this case until the second meiotic division, one nuclear division later than usual, and the deficient microspore so formed would be able to go through the following microspore mitoses although the resulting pollen grain presumably would not be functional since a further division is necessary to produce the male gametes in the pollen tube. Unequal segregation has not been observed at the second meiotic division, but these figures are difficult to analyze and such irregularities should be rare. Unequal distribution of chromosomes at the microspore division has been observed by Sax[7] in *Tradescantia* and is indicated by anaphase figures at the division of the gen-

TABLE II. Frequencies of equal and unequal chromosome segregation at the first meiotic division in *Tradescantia*.

Plant No.	Chromosome distribution 6/6	7/5	
346	100	9 = 8%	unequal divisions
"Harvard"	76	13 = 14.6%	unequal divisions

TABLE III. Frequencies of normal and aneuploid microspores of tetraploid *Tradescantias*. *n* = 6.

Frequencies of microspores			
2n—2	*2n*—1	*2n*	*2n*+1
2	16	171	11

erative nucleus in the pollen tube in rare cases (Sax, unpublished). Such unequal segregation at the microspore division must be rare, however, because no *n*—1 figures were found in about 5,000 generative nucleus divisions in *Tradescantia* pollen tubes (Swanson, unpublished).

The results obtained in *Tradescantia* indicate that monosomic plants are not produced by meiotic irregularities in pollen mother cells. Functional deficient gametes could be produced by non-disjunction in the microspore division or in the division of the generative nucleus in the pollen tube. Somatic non-disjunction in the sporophytic tissue would result in monosomics, particularly the chimeral types, and probably is the most important factor in the production of monosomic plants. It is possible that unequal segregation of the chromosomes in the megaspore mother cell might produce a functional *n*—1 egg cell since the female gametophyte includes a large amount of cytoplasm which might be conditioned for a number of cell divisions, as is the case in certain animal eggs, reported by Harvey[3] and Painter.[6]

Although *n*—1 microspores in plants appear to be inviable, *n*—1 sperm can and do function at fertilization.[2,4] The most obvious explanation for this difference is simple. In spermatogenesis there is no nuclear division between meiosis and fertilization and the chromosomes of a deficient sperm cannot divide until after fertilization, when a complete female genom is present to cover the deficiency. In the higher plants there are two divisions in the male gametophyte between meiosis and gamete formation, and it is this factor which inhibits the survival and function of deficient microspores. The relative difference in complexity of the gametophytic development in plants and animals must be of some significance in respect to the survival of certain mutations and chromosomal aberrations.

Summary

Unequal segregation of meiotic chromosomes in diploid *Tradescantia* produces many microspores with more than the haploid chromosome number. The corresponding deficient microspores must be produced in at least equal numbers, but very few of them develop. Evidently deficiencies produced at the first meiotic division in microsporogenesis do not inhibit the second meiotic division, but prevent the development of the microspores. The few *n*—1 microspores observed are attributed to unequal segregation at the second meiotic division. Deficiencies produced at either meiotic division will produce non-functional pollen grains. The elimination of deficiencies in the male gametophyte of plants is contrasted with the viability of deficient sperm in animals.

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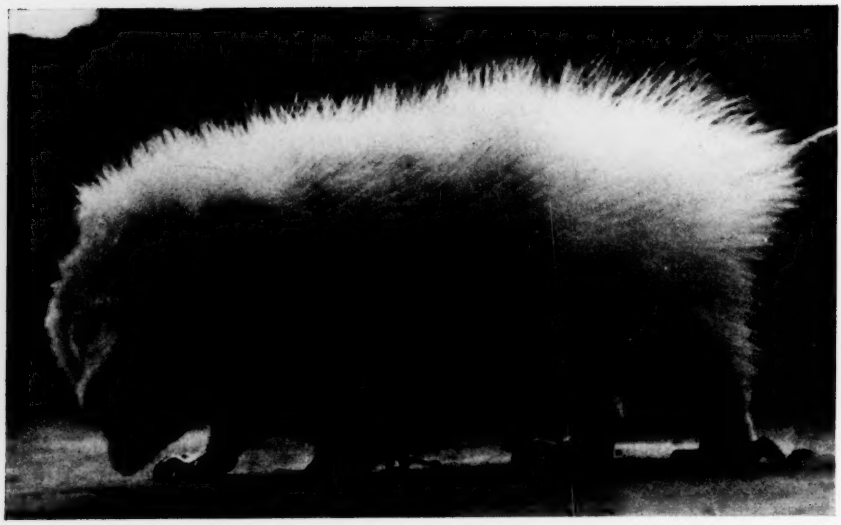
An Albino Buffalo

A small buffalo herd had been introduced by the Biological Survey several years ago into the Big Delta River and Jarvis and Granite Creek region near McCarty, Alaska. During the last 2 years a careful check has been kept on this herd, and the last inventory showed the herd has increased to about 200 animals. Among them is an albino buffalo, one of the three albinos of the species known to be alive in North America.



ALBINISM IN THE OPOSSUM

Figure 1

PIGMENTATION in mammals depends upon the presence and interaction in the tissues of the color-bases, colorless themselves, and ferments or enzymes which act upon the color-bases to yield color products. The pigmentless albino is an individual whose tissues lack the power to elaborate either the ferments or the color-bases.

Albinism has been recorded in a great majority of the species, breeds, and varieties of domesticated animals and cultivated plants. It has been known to occur among squirrels, lobsters, badgers, doves, dogs, robins, frogs, and rodents.

An albino opossum was captured in December, 1939, near Cushing, Oklahoma. "Pinkie" as it is called, weighs 4½ pounds and is about half-grown. Its eyes are pink with apparently no pupil. Its fur is softer than the common opossum's. Its skin and the scales on its prehensile tail are white.

The fur of the ordinary opossum is greyish-brown in color and woolly, consisting of a mixture of long bristle-like hairs and a fine under-fur. Thirteen records of albino opossums have been obtained. About one-third of this number mentioned the softness of the fur. In the other two-thirds, the condition of the fur was not noticed. Information obtained from a Lindsay, Oklahoma, fur dealer stated that the albino opossums he has obtained do not have any true fur at all; that it is all hair.

Albinism probably has an hereditary basis in the opossum similar to that determined for other vertebrates, wherein true albinism is universally a recessive. The peculiar hair-like condition of the fur noted in the opossum appears to be a unique and hitherto unnoticed feature of albinism in this species.

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A NEW MUTATION IN THE MOUSE

Affecting Spinal Column and Urogenital System

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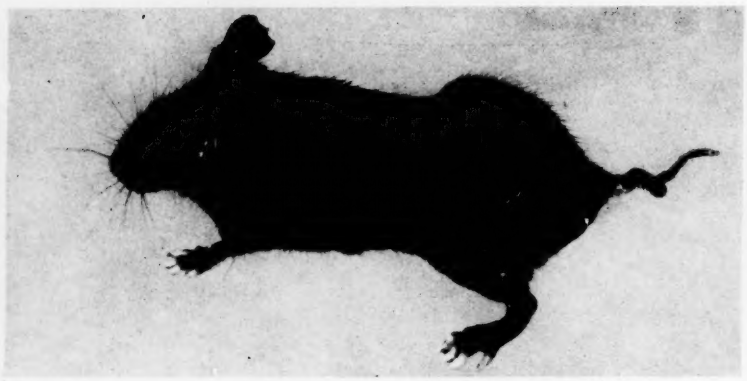
THE tail is so prominent a feature of the laboratory mouse that mutations affecting its structure are especially easy to detect and to study. In recent years six mutations of this sort have been reported, while several others are being tested in this and other laboratories. Each of these mutations affects structures or processes in addition to those localized in the tail and it is often these other effects which give the mutation its chief interest. Thus the short-tail (Brachyury) mutation, *T*, proved to effect early morphogenetic processes involving the notochord and somites,[1] while two other mutations at or near this locus (t,[9] t^1) similarly produce profound changes in the early embryo,[2,3] have an early lethal effect, and modify as well the segregation ratios in males heterozygous for them.[4] The recessive mutation "flexed tail" causes a change in the differentiation of intervertebral cartilages and produces an embryonic and juvenile anaemia[5] while the mutation known as shaker short[6] modifies not only the development of the end of the tail but alters the growth of the brain and the internal ear and results in a radical departure from the normal pattern of behavior.[7] Observations on other effects of "fused" and "kink" tail mutations have not been completely published but they are profound and widespread. While it is thus not surprising to find that the tail mutation most recently reported[8] also has far reaching effects, the fact that these include the important condition known as spina bifida and the partial or complete suppression of development of the kidneys and other parts of the urinary and genital systems lends a special interest to this case. In this paper the evidence on the gross effects, the inheritance and some of the interaction relations of the new mutation will be described. The results of an anatomical and embryological study will be published elsewhere.

The mutation was first found by Professor C. H. Danforth among descendants of his "posterior duplication" stock.[9] He kindly sent to us in October 1936 several short-tailed animals which appeared to him to resemble the Brachyury mutation of Zawadskaia which we were studying. Animals with the new mutation had tails varying from a short stump to about 1/3 of normal length. Such short-tailed mice when inbred in our laboratory and when crossed to normal and to Brachy stocks proved to contain a new dominant mutation to which we gave the symbol *Sd* (short-Danforth). Our stocks of this mutation are all descended from four mutants (two males and two females) received from Prof. Danforth. They apparently do not contain the gene complex "posterior duplication" which had been present in the ancestral stock.

Description of Mutant

We soon found that the heterozygous mutant phenotype is somewhat variable, although always easily distinguishable from normal. It has either no tail at all, or a short non-bony filament of skin and connective tissue (the remains of a tail which was present in the embryo) or a short tail never exceeding about one half the normal length and usually terminating in a short contorted filament. For brevity's sake we shall refer to this phenotype as short or *Sd*. including in this term both short and tailless. The sacral region of such mice frequently appears shortened, due to malformations in the sacral vertebrae, and there are occasionally crooks in the spine (scoliosis or



A SHORT-TAILED MOUSE

Figure 2

Adult mouse showing the new short-tail mutation (*Sd*). The base of the tail has vertebrae, but the twisted tip is merely a filament of skin and connective tissue, which in some short-tailed mice fails to persist, leaving a blunt end.

lordosis). In these heterozygotes the kidneys are usually abnormal, either one or both are missing or they are abnormally small.

Segregation Ratios

All such *Sd* mutants proved to be heterozygous and when crossed with normals (mostly from the Bagg albino inbred stock) gave about equal numbers of normals and mutants (Table I). The mutant class consisted of short-tailed and tailless phenotypes, and comprised slightly (but not significantly) less than half of the total. Records reported from this and all other crosses are based on observations made shortly after birth, at nine o'clock each morning. All litters were born in isolation pens.

When bred together the *Sd* mutants produce three easily distinguishable phenotypes in a simple ratio: about ¼ of the progeny are normal, about ½ are short like the parents and about ¼ are completely tailless and otherwise abnormal and always die within 24 hours after birth. The latter are readily recognized and are undoubtedly the homozygous mutant class *Sd Sd*. The numbers of these three types obtained (Table I, line 1) fit a 1:2:1 ratio, although the number of heterozygotes is somewhat high. This is due to the offspring of one exceptional male which gave 7 normals, 40 "short" and 11 homozygotes. The reasons for this aberrant ratio are not apparent. One son was tested and gave normal ratios. When the aberrant ratio is subtracted the total becomes 152 normal:328 heterozygotes:142 homozygotes, corresponding to a 1:2:1 ratio.

Homozygotes

The homozygotes show a very interesting syndrome of pathological characters. They are entirely tailless; they have either no genital papilla or an inconspicuous one so that it is impossible to distinguish the sexes among them; and there is always an imperforate anus. In the sacral region there is frequently a lesion under the skin, a blood bleb or hematoma as a symptom of spina bifida. Upon dissection or clearing it is found that the spinal column is in all cases extremely short; it generally ends in the lumbar region and in most cases all vertebrae posterior to the second lumbar are missing. The kidneys are entirely absent and there is a genuine cloaca, recalling a more primitive vertebrate condition. Bladder and urethra are present in some homozygotes and missing in others. In spite of these multiple deform-

EARLY STAGE IN DEVELOPMENT OF SHORT TAIL

Figure 3

A twelve-day embryo (with membranes and umbilical vessels attached) showing an early stage of the short-tail mutation. The circulation at the end of the tail has broken down producing a large bleb or haematoma. The shortness of the tail results from the ensuing degeneration of the distal end.

ities this class survives to birth with a viability practically equal to normal siblings, begins to suckle in competition with more normal litter mates, and dies probably from auto-intoxication usually within 18 hours after birth. None has ever survived bevond 24 hours.

Photographs showing skeletons of heterozygous and homozygous *Sd* mice are shown in Figure 4. In Figure 3 is shown a heterozygous embryo which is destined to be short-tailed. The mutation causes a circulatory breakdown and the end of the tail then degenerates.

Tail Length of Heterozygotes in Outcrosses

In order to make the residual heredity of this mutant stock comparable with stocks of other tail mutations maintained in our laboratory we undertook systematic backcrosses of heterozygous mutants to the long inbred Bagg albino stock. The effects of this backcrossing on tail length are shown in Figure 5. Stock animals were bred together, this constituting the P_1 generation. These were crossed to Bagg albino without selection for tail length, and the heterozygotes were inbred for two generations (F_1-F_3); from these, heterozygotes were chosen (without selection) backcrossed to Bagg albino and this process repeated through four subsequent backcrosses. As the proportion of Bagg genes increased with continued backcrossing, the tail length of the heterozygotes decreased. The best objective measure of this is the proportion of tailless among all heterozygotes which has now reached nearly 90%. The tail length of the few heterozygotes which have any tails at all does not now exceed ⅛ of the normal length. The conclusion to be drawn is clear: the genes of the Bagg normal-tailed stock increase the expression and thus the dominance of the *Sd* mutation. This is the more surprising since the genes of the Bagg stock are known to have the opposite effect upon the Brachy short-tail

HETEROZYGOUS AND HOMOZYGOUS FOR *Sd* GENE

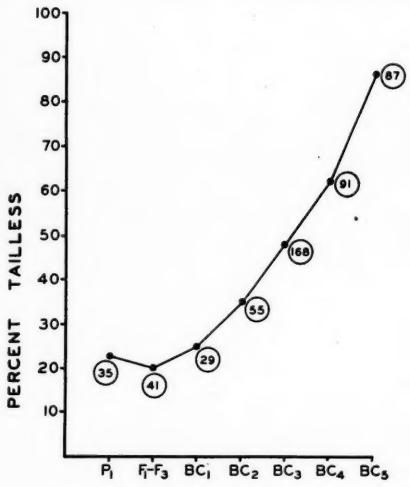
Figure 4

Photographs of cleared and stained skeletons of new-born mice. At the left a heterozygous short-tailed animal in which the vertebral column extends slightly beyond the pelvis. At the right a tailless homozygote in which the vertebral column ends in the upper lumbar region. The specimen at the right showed spina bifida and had no kidneys. All such homozygotes die on the day of birth.

mutation, *T*. The significance of this apparently paradoxical situation will be discussed elsewhere. Here it need only be said that the clue to the differing interaction of *T* and of *Sd* with the genes of the Bagg stock is probably to be found in the difference in the morphogenetic channels through which *T* and *Sd* produce their tail-shortening effects.

Effect of *Sd* on Viability in Heterozygotes

Concomitant with a decrease in tail length of heterozygotes we noticed also an apparent decrease in viability. After this was noticed we tabulated the proportion of heterozygotes which died at various five-day intervals after birth. This led to the surprising result shown in Table II, namely that nearly 70% of all heterozygotes die before weaning with a very heavy mortality between five and ten days of age. Much of this is probably due to the effect of *Sd* on the urogenital system, but some of course is due to the residual mortality common to young mice in our colony. The mortality of the *Sd* heterozygotes is much higher than the residual mortality, and indicates that *Sd* has a dominant effect



INCREASE IN DOMINANCE OF *Sd* GENE

Figure 5

Frequency of tailless young among *Sd* heterozygotes of different backcross generations to Bagg albino. Figures in circles represent total number of animals measured.

on viability as well as on visible characters. Whether its dominance in this respect has increased we are unable to say in the absence of accurate mortality records on the stock when first received. It is our impression however that *Sd* heterozygotes have lower viability when combined with the Bagg albino gene complex. Its behavior in this combination is now approaching that of a dominant lethal and it threatens thus to become extinct. The *Sd* mutation is therefore being bred into other stocks in some of which both tail length and viability show signs of increasing.

Linkage Tests of *Sd* with Brachyury *T*

The new dominant was tested for interaction effects and linkage with the Brachyury mutation *T*. Table III shows the data obtained in the first cross (carried out before *Sd* had been extensively backcrossed to Bagg albino). The results indicate that about equal numbers of normal, Brachy, *Sd*, and tailless were obtained indicating that the double heterozygote of *Sd* and *T* is tailless and thus that these two tail shortening mutations have a cumulative effect. It is unlikely that separation of genotypes in the above cross was accurately made by this phenotypic classification. There was probably some overlapping between *T*-short and *Sd*-short and between *Sd*-short and tailless.

TABLE I.—Results of Matings of "Short"-tail *Sd* Mice

	Normal	Short tail (including tailless)	Tailless with lethal syndrome	Total
Short tail (*Sd*) × short tail (*Sd*)	159	368	153	680
Short ♂ × normal ♀	704	686	.....	1,390
Short ♀ × normal ♂	195	171	.....	366
Short × normal Total	899	857	.....	1,756
		p>0.3		

TABLE II.—Mortality of *Sd*+ after Birth (Based on Observation of 257 Cases)

Age	Daily death rate (Percent)	Total of deaths (Percent)
to 3 days	12	12
3- 5 days	16	28
5-10 days	20	48
10-15 days	10	58
15-20 days	8	66
20-25 days	3	69
25-30 days	3	72

TABLE III.—Independent assortment of *Sd* and *T*. F_1 from crosses of *Sd* by Brachy

Type of cross	Normal	Short "T"-type	Short "Sd"-type	Tailless
♀ *T*+ × ♂ +*Sd*	69	65	54	64
♀ +*Sd* × ♂ *T*+	53	45	38	41
Total: *Sd*+×*T*+	122	110	92	103
Expected	107.25	107.25	107.25	107.25
				p=0.23
10 F_1 tailless (*Sd*/+ *T*/+)× normal	106	97	76	94
Expected	92.75	92.75	92.75	92.75
				p=0.18
7 F_1 tailless (*Sd*+) × normal	93	....	81	....
Expected	87	....	87	....

TABLE IV.—Linkage Test of *Sd* and Caracul

Cross	Normal ++	Caracul *Ca*	Short tail *Sd*	Caracul Short tail *Ca Sd*
+/*Sd*+/*Ca*×++	50	33	35	43
				p>0.2
Expected (random assortment)	40.25	40.25	40.25	40.25

Seventeen of the F_1 tailless animals were tested by crossing to normal. Ten of these produced four classes of young in about equal numbers (Table III, line 5) and were thus double heterozygotes ($Sd+T+$). The ratio obtained indicates independent assortment of *Sd* and *T*.

Seven of the F_1 tailless when tested by normal gave no Brachy type short-tails and proved to segregate only for *Sd* and normal.

The new dominant was tested also for linkage with Caracul (*Ca*), a dominant which can be accurately scored at birth.[10] Table IV indicates that no close linkage exists between *Sd* and *Ca*.

Summary

Mice heterozygous for a new mutation, *Sd*, have either short tails or no tails at all, often shortened or crooked spines, abnormalities of the kidneys and a lowered viability after birth.

Animals homozygous for this mutation show a striking syndrome of abnormalities including complete taillessness, spina bifida, absence of kidneys, of external genitalia and other portions of the urogenital system, and always die shortly after birth. Breeding data show that the whole complex of characters segregates as a unit and does not affect viability before birth.

The expression of the mutation in the heterozygote was enhanced by the genetic constitution of an inbred normal stock (Bagg albino). The viability of the heterozygote has now fallen so low that it is in this stock virtually a dominant lethal.

It is probably not linked with a similar short tail mutation T, nor with Caracul (*Ca*).

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Causes of Early Death

THIS is a survey of the causes of fetal and neonatal death. Included are statistics on still births and on infant mortality in the United States. There are short descriptions of the normal conditions at birth of the various organs and systems and of pathological deviations. Some notes on methods of autopsy are given. The book will be useful to those who desire a mere outline of the subject.

W. Landauer

*Potter, E. L. and F. L. Adair. *Fetal and neonatal death.* XV, 207 p., 31 figs. Price $1.50. University of Chicago Press, Chicago, Ill.

CYTOGENETICAL ANALYSIS OF HETEROSIS IN HYBRIDS OF VICIA

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"The most promising organization of experiments demands a systematic cultivation of races, and, above all, their crossing, combined with an investigation of chromatin in the objects under observation."—BOVERY, 1904.

ON the strength of a number of new experiments and of facts recently discovered in this field, the organization of experiments proposed by Bovery thirty years ago acquires a still greater importance: (1) The study of geographical races has opened quite new aspects of interracial crossings; (2) new and more detailed methods of studying caryotypes give a possibility of discovering differences not only between different species but even between closely related forms. Only a study of differences between species based on the combined cytogenetic method, a thorough analysis of changes in the caryotype of closely related species, and of crosses between them, and the discovering of their linkage groups (for the purpose of tracing chromosome maps)—can be considered as the fundamental method of throwing light on the evolutionary process by which the forms under question have evolved. In combination with Painter's method of studying chromosomes in salivary glands, the cytogenetic method became as essential and acquired the same degree of exactness as the genetic one. It can be said that chromosomal changes, when suitable material is available, furnish finer and more precise morphological indexes not only of the position of a given form in a system but also of those processes which take place within this system.

As yet the problem of heterosis has not been subjected to cytogenetic analysis in parallel with the genetic approach. The present work is an attempt in this direction.

Heterosis in Hybrids of Vicia

Livesay,[22] in summing up the phenomena of heterosis, closes with the following question: "Did an organism ever exist the crossings of which produced a lessening of vigor, or could such an effect be obtained artificially? Such a phenomenon would explain heterosis, the experimental evidences of which are most desirable."

Such a proof, we find, for instance, in hybrids of vetches. By crossing two closely related species, dwarf plants have been obtained—a clear demonstration of minus-heterosis.

In comparing the above facts with our observations on the hybrids of *Vicia*, we have arrived at the following fundamental propositions: (1) it is urgent to extend the concept of heterosis in order that it may explain not only a more powerful development of the organism but a weaker one as well; (2) it is necessary to consider the phenomenon of heterosis as caused by certain combinations of genes.

The material relating to the hybrids of *Vicia* has been analyzed for the purpose of studying heterosis in three different ways:

(a) Dominance of factors which produce a more powerful development:

$AA \times aa = Aa.$

(b) Accumulated manifestation of characters:

$A \times B$ = plus-heterosis.
$A \times C$ = minus-heterosis.

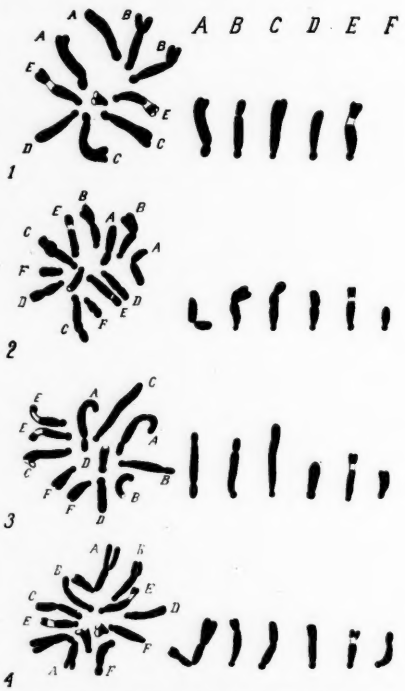
(c) Influence exercised by a partial sterility, due to a certain definite combination of genes, upon the development of a larger vegetative mass.

Differing from the previous works on the causes of heterosis, our investigation was based on material collected to illuminate a definite system of changes in the carotype.

The conspecies *V. sativa* embraces *V. amphicarpa, V. sativa* and *V. angustifolia*. It is difficult to find a principle which could be taken for a standard in judging whether the forms composing the *V. sativa* group are species or varieties, since the concepts of species and variety are as yet not sufficiently defined by the systematists. Schmalhausen,[27] Boissier,[1] and Koch[21] take *V. sativa* and *V. angustifolia* to be independent species. Ascherson (1916) considers them as varieties. These different opinions point however to a close relationship between the forms under question. The recent findings in the morphology of *Vicia*, obtained by Toupikova[36] speak in favor of the assumption that *V. sativa* and *V. angustifolia* are two independent species. Our analysis of the chromosomes corroborates that view.

A description of the conspecies *V. sativa* can possibly, in order to ascertain the phylogeny of forms, be the most easily given if it is based upon such a simple and constant character as the form of chromosomes. We divide the chromosomes of species closely related to *V. sativa* into two groups: instable and stable ones. The first group with the chromosomes *A, E* and *F* (Figure 6) is characterized by its clearly pronounced change in the course of evolution of the vetches. As to the second group of chromosomes: *B, C* and *D* (Figure 6) we did not succeed in discovering any consistent visible changes. On the strength of a number of experimental findings[35] the author and Belekhova hold that a definite statement may be made regarding the origin of *V. sativa* and of the wild forms related to it.

The changes of the characters in *Vicia* are conventionally indicated in the table by the letters of alphabet. "In accordance with the theory of multiple factors, it can be assumed that, in order to define any quantitative character, we must also consider a great number of independent and non-dominant factors which have however no different visible expression, but influence the same character, in this case a quantitative one, their influence accumulating through a simple increase



CHROMOSOMES OF VETCH SPECIES AND RACES

Figure 6

(1) *Vicia amphicarpa* — 10 chromosomes; (2) *V. sativa*—12 chromosomes; (3) *V. angustifolia brachisomica*—12 chromosomes; (4) *V. angustifolia dolichosomica* — 12 chromosomes. At the left are shown metaphase plates of somatic divisions. At the right representative unpaired chromosomes arranged in order. In these closely related species and varieties definite morphological differences can be related to differences in the structure of the chromosomes. See Text, page 351.

or decrease of the magnitude of this character, by a definite quantity" (Sinnot and Dunn, 1932).

The fundamental idea upon which our concept of changes in the characters is based, is a demonstration of a connection between a gradual change in the length of chromosomes in related species of *Vicia* and the parallel growth of definite quantitative characters, which can be explained by the existence of a reduplication of factors due to the dupli-

TABLE I. Changes in the characters of the conspecies of *V. sativa*. Change in the length of chromosome is represented by a fraction; in the numerator — the upper arm, and in the denominator — the lower arm.

	Changes in characters parallel to changes in *A* and *F* chromosomes			Changes in the length of chromosomes			Changes in characters corresponding to changes in chromosome *E*		
	Lower branching	Degree of backbend in standard and wings	Number of flowers per raceme	Length of chromosome *A*	Length of chromosome *F*	Length of chromosome *E*	Branching along the stem	Size of leaflet	Height of plants
V. amphicarpa	M_1M_1	O_1O_1	N_1N_1	$\frac{A_1A_1}{A_1A_1}$	.....	$E_1E_1F_2F_2$	H_1H_1	$I_1I_1I_2I_2$	PP
V. sativa	$M_1M_1M_2M_2$	$O_1O_1O_2O_2$	$N_1N_1N_2N_2$	$\frac{A_1A_1}{A_1A_1A_2A_2}$	F_1F_1	$\frac{E_1E_1F_2}{F_2E_3E_3}$	$H_1H_1H_2H_2$	$I_1I_1I_2$ $I_2I_3I_3$	$P_1P_1P_2$ $P_2P_3P_3$
V. angustifolia brachisomica Race *A*	$M_1M_1M_2M_2$ M_3M_3	$O_1O_1O_2O_2$	$N_1N_1N_2N_2$	$\frac{A_1A_1A_2A_2}{A_1A_1A_3A_3}$	$F_1F_1F_2F_2$	E_1E_1	H_1H_1	I_1I_1	P_1P_1
V. angustifolia brachisomica Race *B*	"	"	"	"	"	"	"	"	P_2P_2
V. angustifolia dolichosomica	$M_1M_1M_2M_2$ $M_3M_3M_4M_4$	$O_1O_1O_2O_2$ O_3O_3	$N_1N_1N_2N_2$ N_3N_3	$\frac{A_1A_1A_2A_2}{A_1A_1A_2A_2A_3A_3}$	$\frac{F_1F_1F_2}{F_2F_3F_3}$	E_1E_1	H_1H_1	I_1I_1	P_4P_4

cation of parts of chromosomes. A comparative description of the species, given here, is based on chromosomes *A*, *E* and *F*, and six characters conventionally represented by the following alphabetical symbols:

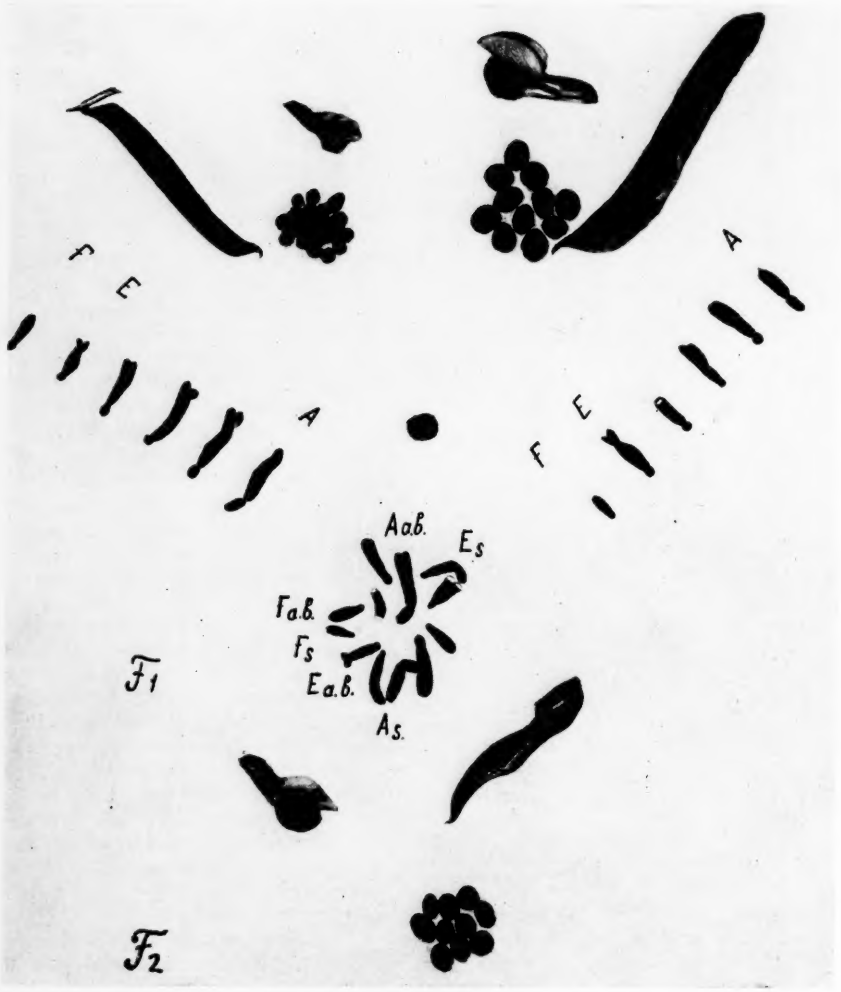
1. Branching beginning at the base ..... *M*.
2. Size of the leaf ..... *I*.
3. Height ..... *P*.
4. Branching the length of the stem ..... *H*.
5. Number of flowers per raceme ..... *N*.
6. Degree of the backbend of standard and wings ..... *O*.

The presence of homologous chromosomes in the group of nearly related species of *V. sativa* is one of the principal facts ascertained through cytological investigations of caryotypes and reductional division in the interspecific hybrids of *Vicia*.

In order to give a comparative description of this group it must be said that the phylogeny of the nucleus in the varieties studied can be clearly traced by the chromosomes; in all four representatives of the group we can trace a gradual change in the homologous chromosomes. This is particularly easy owing to the close relationship of the group, — a relationship so near that it makes us doubt whether they are species or varieties. The question of the direction of the evolutionary process—whether there has been a shortening or a lengthening of chromosome arms — remains, as in other similar cases, open.

Let us trace on the table the behaviour of separate characters in connection with the changes in chromosomes. Three characters (1) basal branching, (2) degree of bending back of standard wings, (3) number of flowers — all change progressively with the

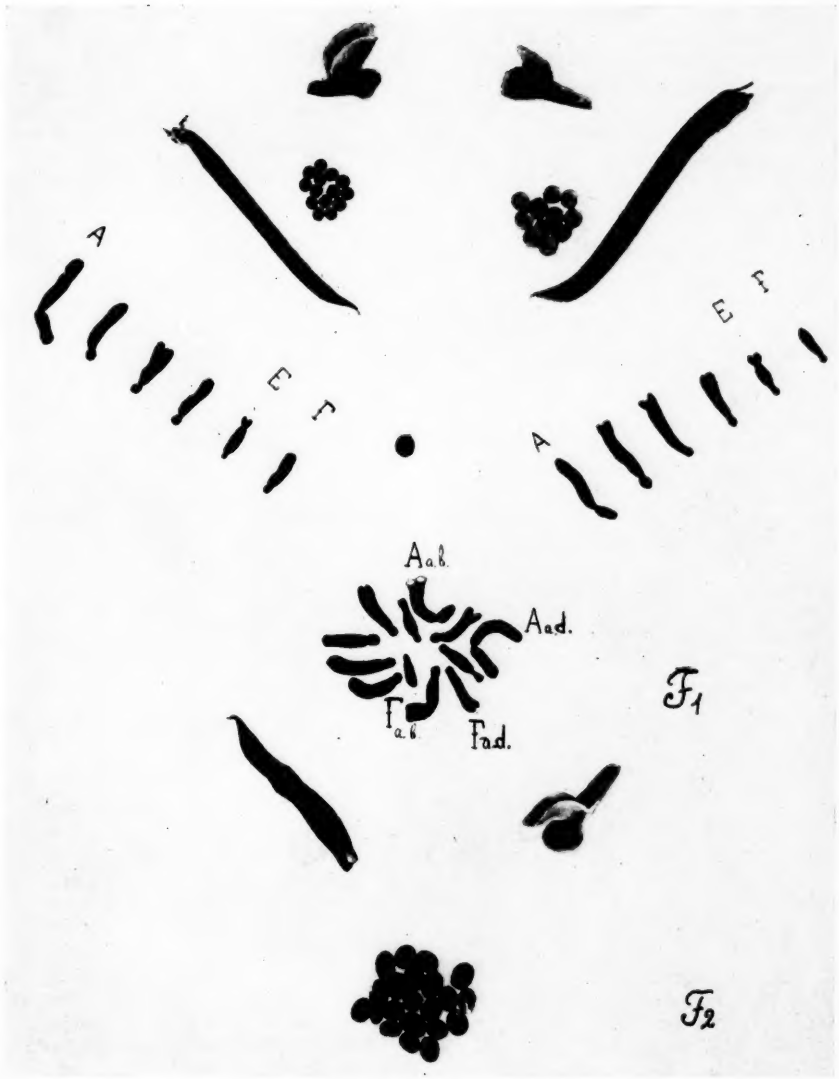
V. sativa *V. augustifolia brachisomica*



A SPECIES HYBRID

Figure 7

Pods, beans, flowers and chromosomes of the cross *V. sativa* × *V. angustifolia brachisomica*. The chromosomes are identified by letters referring to Figure 6. The chromosomes in the F_1 are identified by subscript letters (s = sativa).



A CROSS BETWEEN RACES DIFFERING IN CHROMOSOME LENGTH

Figure 8

Pods, beans, flowers and chromosomes of *V. angustifolia brachisomica* × *V. angustifolia dolichosomica*. The F_1 hybrid is partially sterile, and extremely vigorous, in spite of the close relationship of the parents.

change in the *A*- and *F*-chromosomes.

Each character changes according to the following scheme. The *A*-chromosome in *V. amphicarpa* has no arms, but its head is larger than that of the other chromosomes. In *V. sativa*, the *A*-chromosome has already two arms. In *V. angustifolia brachisomica*, one of the arms, and in *V. angustifolia dolichosomica*—both arms become longer. The *F*-chromosome is absent in *V. amphicarpa* and appears in *V. sativa*, gradually increasing in size in the following species (see Table I). The basal branching of the plant becomes progressively more extensive, beginning with 2-3 branches in *V. amphicarpa* and mounting to as high as 20 branches in *V. angustifolia dolichosomica;* the standard bends back very slightly in *V. amphicarpa*, quite fully—in *V. sativa*, and in *V. angustifolia dolichosomica*, not only the standard, but the wings as well, are bent back. The quantity of flowers per raceme increases from one in *V. amphicarpa* up to three in *V. angustifolia dolichosomica.*

The *E*-chromosome with three limbs undergoes a change somewhat different from the changes in the *A*- and *F*-chromosomes. Its third articulation, short in *V. amphicarpa*, grows longer in *V. sativa;* the whole chromosome decreases strikingly in size in *V. angustifolia*, but keeps its characteristic form with three limbs. The characters: height of the plant, extent of basal branching, length of the stem and size of the leaf—present analogous phenomena.*

The Hybrids

Crosses have been effected between the above described species of the system under discussion and about 500 plants of the first generation have been obtained.

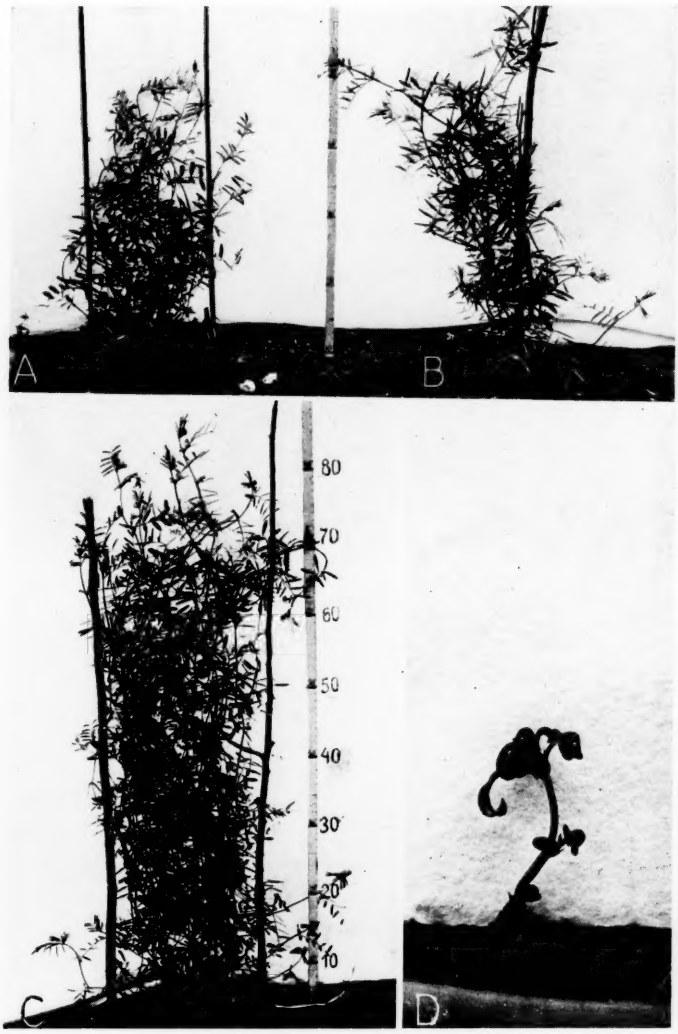
All the hybrids of the first generation have given evidence of an exceptionally strong heterosis which has been partially inherited by the offspring. Three combinations of crosses have proved to be of the greatest interest in this connection::

1. *V. sativa* × *V. ang. brachisomica.*
2. *V. sativa* × *V. ang. dolichosomica.*
3. *V. ang. brachisomica* × *V. ang. dolichisomica.*

V. angustifolia presents a highly polymorphic species; from among this multitude of forms, one form with a lengthened *A*-chromosome has been found, and named accordingly "dolichosomica" (Figure 6*D*). As Figure 8 shows, *V. angustifolia dolichosomica* differs from *V. angustifolia brachisomica* only by its narrow black beans and uniform seeds, the smallest of the vetches. This form has no varieties, differing thereby from the polymorphic species *V. sativa* and *V. angustifolia brachisomica.* The hybrid of *V. angust. brachisomica* and *V. angust. dolichosomica* has beans of irregular form (Figure 8), owing to a partial sterility of the plant. The height of the plant exceeds that of the parent plant by 150 per cent on the average (Figure 9 *A-C*). We have here a case of plus-heterosis which can be explained by a cumulative manifestation of the factors of growth P_1P_1 in *Vicia angustifolia brachisomica* and P_4P_4 in *Vicia angustifolia dolichosomica.*

The author[32] thus summarized this situation in 1929: "Therefore, we consider the characters of the nucleus, the form of the chromosomes as a certain constant value, and its difference in the two forms clearly shows that an essential process has taken place which has divided these two forms and made them into two independent units. Guided by this character, we can, for instance, affirm that all the diversity of form in *V. angustifolia* is peculiar to the one of the units—*V. angust. brachisomica*, and that the race *V. angust. dolichosomica* is another particular unit which produces, if crossed with *V. angust. brachisomica,*

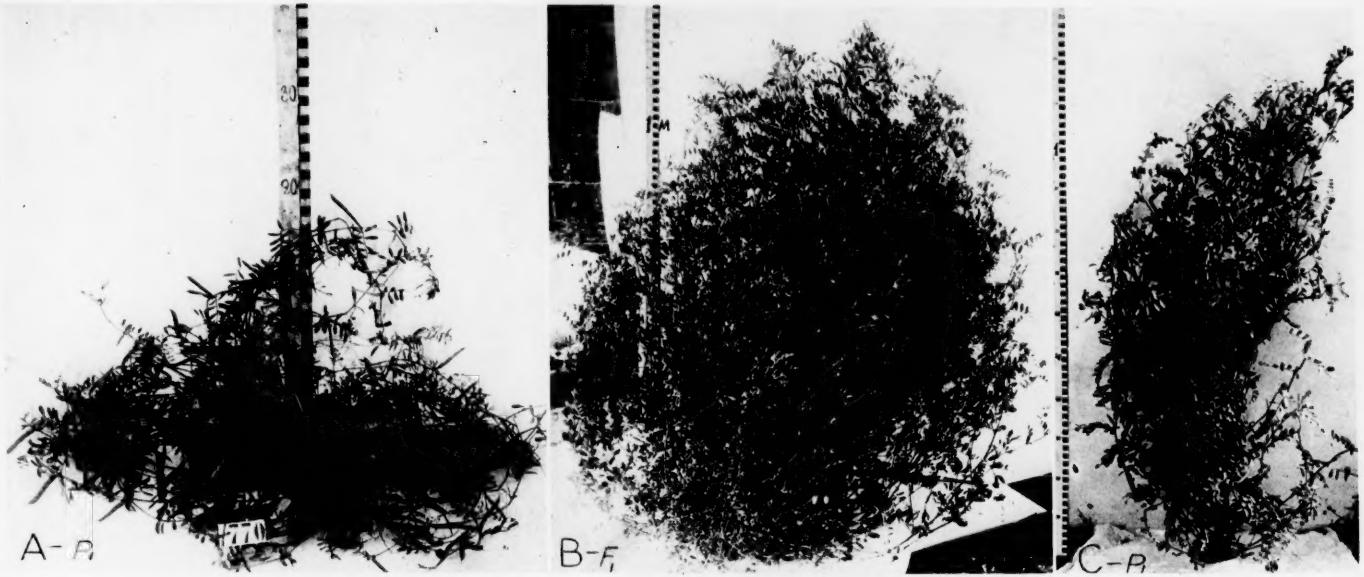
*The initial material has been procured from the unique world collection of Vicia made by A. J. Toupikova (State Institute of Plant Industry). For the purpose of cytologic analysis of the somatic and of the reductional division, the hybrids have been fixed by the method of Navashin (10:4:1) and Lewitsky (5:5). The staining of the preparation was effected by means of haematoxilin, according to Heidenhain.



PLUS AND MINUS HETEROSIS

Figure 9

The sub-species *dolichosomica* (*A*) of *V. angustifolia* differs from *brachisomica* (*B*) only in having narrower leaves and smaller seeds. Remarkable differences in heterosis are encountered in hybrids between *dolichosomica* and two races of *brachisomica*. With one, the hybrid (*C*) shows marked plus-heterosis, being fully 150 per cent larger and more branched than either parent. With the second sub-race of *brachisomica* the hybrids are dwarfs, only a few centimeters high (*D*). Eight such dwarfs have been obtained. All but one was entirely sterile.



EXTREME HYBRID VIGOR IN SECOND GENERATION

Figure 10

A — *V. angustifolia; B* — F_2 segregate; *C* — *V. sativa.* Here in the second generation an extreme example of heterosis is to be found. Usually heterosis is more marked in the first generation, and largely disappears in later generations, but in this cross some second generation segregates exceeded the vigor of the first generation.

VARYING HETEROSIS

Figure 11

At the left is a plant of *V. angustifolia dolichosomica*, at the extreme right, a plant of *V. sativa*. Between them are second generation hybrid plants showing varying degrees of "negative heterosis" — none of these plants equaling in hybrid vigor either of the parent species. About a third of the plants in this cross died in the seedling stage.

a remarkably expressed picture of heterosis; this picture supports, on the one hand, the assumption of the close relationship of these forms, and, on the other hand, that of a dissimilarity of the genes of growth in these two botanical units." Among the various races of *V. angust. brachisomica*, there is still one form greatly differentiated, which we are analyzing cytogenetically, and which will presumably be found likewise different as to the morphology of its chromosomes. This race has unusually narrow and long leaves and a rather thick stem; it has always but one single flower per raceme. Crosses of this race of *V. angust. brachisomica* with *V. angust. dolichosomica* produced plants a few centimeters high (Figure 9*D*). One of eight such dwarfed hybrids F_1, yielded two seeds. The seven remaining plants were absolutely sterile, and did not even bloom. Such dwarfs might possibly be haploids, but a cytological analysis shows that our plants had the normal diploid number of chromosomes—12. The plants showed an unusually depressed development, a small height and small crinkled leaves. We find here for the first time in the first generation hybrids a clearly pronounced phenomenon of depressed development, which may be called minus-heterosis. The occurrence of dwarfed plants when the hybridization is remote, is usually explained by incompatibility due to their great phylogenetic differences in the cross. The cause of minus-heterosis lies here not in a remote relationship of the parental forms, since they are but different forms of the same species, *V. angust. brachisomica*. The cause of depressed development appears here to lie in a definite combination of genes; it can be supposed (see Table 1) that P_2P_2 are derived from *V. angust. brachisomica* race *B* and P_4P_4 from *V. angust. dolichosomica*.

Another explanation is also possible.

The *B* race of *V. angust. brachisomica* may possess a dominant factor (*DD*) for depressed growth, which is balanced by another factor (*KK*). *V. angust. dolichosomica* possesses correspondingly *dd kk*. In the F_1 we have *DdKk* with a clearly pronounced dwarfing of the hybrid. This fact particularly corroborates the above described observation that plus-heterosis is produced by crosses between *V. angust. dolichosomica* and the other race—*V. angust. brachisomica*. It is obvious that the cause lies here not in a different number of heterozygous elements, since these two races of *V. angust. brachisomica*, which have been crossed with *V. angust. dolichosomica*, are very nearly related; the cause is evidently to be looked for in a certain combination of the "genes of growth."

It is to be pointed out that this case was not unique,—other crosses support our deductions, namely: *V. sativa* × *V. angust. brachisomica* (Figure 7) and *V. sativa* × *V. angust. dolichosomica*. As regards the first generation of these hybrids, we must note a powerful manifestation of plus-heterosis due to the cumulative action of a small number of multiple factors of the three dominant characters: basic branching $M_1M_1M_2M_2$ M_3M_3 in *V. angustifolia*, height P_1P_1 $P_2P_2P_3P_3$, branching along the stem $H_1H_1H_2H_2$ (in *V. sativa*) (Figures 10 and 11). Dominant characters were those of species possessing lengthened chromosomes and having at the same time strongly pronounced quantitative characters, which we have conventionally indicated by a large set of factors (see Table I). In 1929, the author has already noted the "dominance (among hybrids) of plants with comparatively long chromosomes.[33] The later observations[35] on the dominance in hybrids of the conspecies *V. sativa* confirm this hypothesis: the longer the chromosomes, the more dominant is the general habitus of a given species, particularly a stronger branching which produces thus a strongly pronounced heterosis in the hybrids.

The increasing of the length of the chromosomes *A* and *F*, accompanied by the parallel rise in the number of quantitative characters and the dominance of parental characters, including longer chromosomes in the hybrids — can be explained by translocations which tend to duplicate parts of chromosomes with definite plural factors.

In the second generation, the hybrids *V. sativa* × *V. angust. brachisomica* and *V. sativa* × *V. angust. dolichosomica* behaved quite differently, according to which form of *V. angustifolia* had been crossed with *V. sativa*. Crosses between *V. sativa* and *V. angust. dolichosomica* produced F_2 plants which were partly etiolated. Six per cent of the seedlings in this cross were so greatly lacking in chlorophyll that they perished after a few days. The remaining plants developed normally the first fortnight. After that, the ends of leaves in one-fourth of the hybrids began to wither (a phenomenon which has also been observed in some hybrids of wheat). The plants became pale yellow, ceased to grow, and did not bloom (Figure 11). The remaining 70 per cent of plants presented a transition from full normal growth to the etiolated state. These facts can be explained by a dihybrid scheme of splitting. Let us call the factors of the presence of chlorophyll in *V. sativa RR*, and the absence of the same by the recessive *rr*, and correspondingly in *V. angustifolia* by *KKRR*. By crossing *V. sativa* (*KKrr*) with *V. angust. dolichosomica* (*kkRR*), we obtain the hybrid *KkRr*. In the second generation, we can expect to obtain one sixteenth of plants *kkrr*, which perish soon after sprouting, and four sixteenths which will survive but will remain sick (etiolated), with but one dominant chlorophyll factor *Kkrr* and *kkRr*.

We obtain actually about six per cent of plants perishing very young, and 26 per cent of etiolated plants, but having enough chlorophyll to live and even to bloom. This is obviously the group with one dominant and three recessive factors. Three chimeral plants were produced only by this one cross — a part of the plants were completely etiolated, another part had leaves green striped and with a part of the branches

with normal leaves — obviously a vegetative splitting had taken place.

It is conceivable that a certain particular combination of genes might, in some other cross produce, on the contrary, an unusually powerful heterosis in the second generation. This supposition has been confirmed by another cross of *V. sativa* with *V. angust. brachisomica* which yielded the best material from the selectionist point of view. Vigorous plants obtained by this cross are superior to the plants of the first generation (Figure 10*B*), and generally to all plants obtained from other crosses. We can observe in them all the useful characters of *Vicia* expressed with the maximum vigor: a compact, upright shrub, not easily broken down, with a powerful branching at the foot of the plant as well as from the axils of the upper leaves; soft leaves and stems constituting an enormous vegetative mass. The height of these plants reaches 1.25 meters. It is quite possible but not finally proved, that a third *F*-chromosome is also present which has been produced by an irregular reductional division and by the formation of a ring of four chromosomes. On the strength of the above described regularity in the parallel increase of the branching and the lengthening of the *F-chromosome*, it is possible to infer that, having here a trisomic as to the *F*-chromosome, we have obtained a triple set of the branching factors, namely: 1-$m_1 m_1 m_2 m_2$: 2-$m_1 m_1 m_2 m_2 m_3 m_3$, which gives a picture analogous to that observed in triploids — the leaves of triploid *Vicia* being much larger than those of diploid parents, taken together. The connection becomes thus clear between the powerful development of polyploids and the heterosis of hybrids: in both cases we obtain duplication of definite, but somewhat different, factors. We have seen that the gene of albinism did not manifest itself either in the initial forms of *V. sativa* and *V. angustifolia* nor in their crosses with other species, but asserted itself only in a particular combination of certain genes; the genes of powerful development could similarly lie hidden in both the parents and manifest themselves fully only in the second generation, namely in the given combination.

This hypothesis opens large perspectives before us. Interspecific hybridization acquires an added importance, since we now, in a certain degree, possess the method of fixation of these forms through chromosome doubling with colchicine, high temperatures and in other ways. These hybrid forms are thus on the way to become new species since by making polyploids of them we can fix them. Uniform and vigorous forms of the tobacco plant successfully obtained by Kappert suggest the possibility of fixing the heterosis of hybrids in other objects too: animals as well as plants To effect crosses between *V. sativa* and *V. angust. brachisomica*, to select valuable forms, and to fix them through some of these methods — is now the most important work to be done in the breeding of Vetches.

Conclusions

Examining the above hypotheses concerning the causes of heterosis in the light of the facts observed in Vetch hybrids, the author has been brought to the following conclusions: firstly, the concept of heterosis must be extended; the phenomenon must be described not exclusively as a comparatively powerful development of the organism, i.e., as plus-heterosis, but also as a depressed, and even quite dwarfed development, or as minus-heterosis; secondly it would be reasonable to consider the phenomenon of heterosis as a visible effect produced by a combination of different genes. The latter concept has been studied in various ways.

The results of experiments made in the course of these six years supported by the observations on dominance in interspecific hybrids of *Vicia*, have led the author to conclude that dominance might depend on duplications caused by translocation and that it can manifest itself externally in the lengthening of definite chromosomes. This phenomenon has already been pointed out by the author in

the description of hybrids of the first generation.

The following observations on dominance in the hybrids of *Vicia* confirm the hypothesis: the longer the *A*-chromosome the more dominant is the general habitus of a given species. The power of branching in certain species increases proportionally to the lengthening of the *F*-chromosome and is transmitted to the hybrids as the dominating factor of species with a longer chromosome.

It is evident that, when studying the phenomena of dominance and heterosis, such a simple and constant character as the morphology of chromosomes is an important indicator of a given process.

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Grants for Research in Human Heredity

The National Research Council Committee on Human Heredity has at its disposal certain funds for use in furthering studies in human inheritance. Qualified persons are invited to submit requests for grants where funds are needed to initiate or to continue research in this field. Requests should be sent as soon as possible to the Division of Biology and Agriculture of the National Research Council, Washington, D. C. They should include a clear statement of the problem, results already achieved, if any, the approximate time required to finish the problem, the amount of money needed, and a statement as to how the money will be spent if granted.

Halbert L. Dunn, Bureau of the Census, Washington, D. C.

L. C. Dunn, Department of Zoology, Columbia University.

K. S. Lashley, Department of Psychology, Harvard University.

George L. Streeter, Bureau of Embryology, Carnegie Institution of Washington, Baltimore, Maryland.

Sewall Wright, Department of Zoology, University of Chicago.

Laurence H. Snyder, Chairman, Department of Zoology, Ohio State University, Columbus, Ohio.

HYBRID VIGOR IN MAIZE EMBRYOS*

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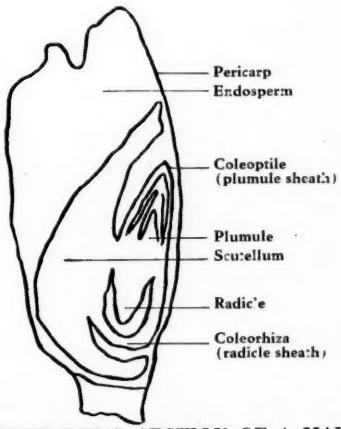
CERTAIN forms of hybrid vigor in corn can readily be detected by mere observation. Thus when the kernels of three strains of corn, i.e. two distantly related inbred strains and their progeny are planted and grown in parallel rows, the hybrid may be seen to be taller than either of its parents throughout the entire growth of the plants. However, in order to determine the presence of hybrid vigor in kernels, size of embryos, size of stomata, etc., accurate measurements are necessary.

In 1930 Ashby[1] presented a new approach in determining hybrid vigor in corn. The writer wishes to point out that the results presented by Ashby do not imply that all crosses of corn showing hybrid vigor would give similar results. The phase of Ashby's work with which the writer is mostly concerned in this article is that dealing with the dry weights of embryos. The difference observed on the part of the hybrid was that of a greater initial weight of the embryo, which gave an advantage that was maintained throughout the entire period of growth. Such a test was carried out by the writer with two different crosses. The hybrid in each cross maintained a noticeable height advantage throughout the growth of the plants. The data concerning the dry weights of embryos are presented in this article.

In 1932 Ashby[2] again presented similar work on other strains of corn and again obtained similar results. By this time several workers were severely questioning his results. The data they presented were far from being in accordance with his. However, a thorough study of the data and comments presented by these workers revealed that a clearer understanding was needed of what was meant by the term, "embryo," as



LONGITUDINAL SECTION OF A MAIZE KERNEL
Figure 12

The coleoptile, plumule and radicle are the parts of the embryo which actually grow and differentiate into the new plant.

used in Ashby's work. The accompanying photographs may help the reader in understanding the following description. (See Figures 12 and 13.)

The writer wished to use Ashby's technique in the dissection of corn embryos. However, even after several hundred had been dissected from eighteen different strains, the data were not at all consistent. Up to this time that part of the embryo which attaches the scutellum to the hypocotyl was partially or completely removed with the scutellum. Ashby does not discuss this part of the dissection. Since he states, (2. p. 1014), that "The scutellum does not undergo cell division, nor does it become part of the adult plant; it was therefore dissected away before the embryo was weighed," the writer deemed it justifiable to remove as much of this attachment tissue as was possible. A razor blade

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accomplished excellent results. Since the root sheath, coleorhiza, does not become a part of the matured plant, the writer also dissected it away. In order to determine the individual dry weights of the embryos, two sets of two distantly related inbreds and their cross were used to put this improved technique to test. Before the dissection of the embryos of the strains (169, 170, and 169 × 170, and 2, 1, and 2 × 1) the kernels were soaked in water for twelve hours at room temperature and then soaked in the same water for twelve hours more at 38° F. The experiment proved quite successful. The individual dry weights of the embryos were amazingly consistent, as will be noted in the following data:

TABLE I. Individual dry weights of embryos in milligrams.

Inbred 169 (used as female parent)	Hybrid 169×170	Inbred 170 (used as male parent)
1.6	2.1	1.9
1.7	2.2	2.0
1.7	2.2	2.0
1.7	2.2	2.0
1.7	2.2	2.1
1.8	2.2	2.1
1.8	2.2	2.2
1.8	2.3	2.3
1.8	2.3	2.3
1.8	2.5	2.3
1.8	2.6	2.3
1.8	2.6	
1.8	2.6	
1.9	2.9	
1.9	3.0	
1.9		
n =17	n =15	n =11
M=1.8	M=2.4	M=2.1
σ =0.087	σ =0.279	σ =0.155
C =4.8%	C =11.5%	C =7.4%

169×170 vs. 170; t=3.2; Odds 1,454:1
170 vs. 169; t=6.6; Odds greater than 185,184:1
169×170 vs. 169; t=8.4

The weights of the kernels of these three strains were also determined. The results were as follows:

Mean weight of 160 kernels in grams.
169=0.246
169×170=0.234
170=0.232

The statistical results of the dry weights of embryos of the other cross are as follows:

DISSECTION OF A MAIZE KERNEL
Figure 13

A—complete kernel; *B*—pericarp and endosperm (Embryo removed); *C*—complete embryo; *D*—scutellum (Embryo axis removed); *E*—embryo axis—upside down; *F*—Coleorhiza root sheath; *G*—embryo axis without root sheath—upside down.

Inbred 2 (used as female parent)	Hybrid 2×1	Inbred 1 (used as male parent)
n =22	n =22	n =17
M=3.0	M=3.2	M=3.0
σ =0.238	σ =0.26	σ =0.26
C =7.9%	C =8.2%	C =8.5%

2×1 vs. 1; t=2.3; Odds 92:1
2×1 vs. 2; t=2.58; Odds 214:1

Weights of kernels. Mean of 160 kernels in grams.
2=0.217
2×1=0.237
1=0.372

An analysis of these data brings out several important points.

1. The hybrid embryos of both

crosses are heavier. Therefore, hybrid vigor is found to be present before germination has taken place.

2. The weights of the embryos of the inbred strains 169, 170, and 169 × 170 have coefficients of variability of only 4.8%, 7.4%, and 11.6% respectively. The coefficients of variability of the strains 2, 1, and 2 × 1 are 7.9%, 8.2%, and 8.5% respectively. If this is found to be true for other inbreds and their crosses, individual weighings should be unnecessary and large numbers of embryos need not be used.

3. The statistical analysis clearly shows that the greater weight of both hybrids over the heaviest inbred is not due to chance. It also shows that the greater weight of the inbred 170 over the other inbred 169 is not due to chance. Student's *t* method was used and the tremendous odds prove that the differences in weights are highly significant.

4. The weight of the kernels shows that the hybrid and its male parent 170 are practically identical, whereas the weight of the female 169 is noticeably heavier than the other two strains. The kernels of the male parent of the other cross are by far the heaviest of the three strains.

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A SORGHUM SEED COLOR CHIMERA

J. B. Sieglinger*

A BI-LATERAL, pericarp-color chimera involving a panicle of sorghum was found at Woodward, Okla., in 1938, in an F_5 progeny row segregating for red and white seed. Nearly half of the panicle (Figure 14) bore seeds which appeared to be white, but which, after threshing, showed variegated red striping on the basal portion of the seed that had been covered by the glumes. The other half of the panicle bore red seeds.

The plant was taken to the greenhouse but it died after the main (chimera) panicle and two axillary branch panicles had matured. The latter arose at the second and third nodes from the top, on opposite sides of the stalk, and were entirely red-seeded.

The seed of most of the branches of the chimeral head was of one color type but a few branches along the border of the sector bore seeds of both types. Seed branches with mostly red kernels bore ten striped seeds and those with mostly striped seed bore 51 red seeds. The panicle produced 920 red and 675 striped kernels. The average weights of the red and striped kernels were 24.6 mg and 26.6 mg, respectively.

In 1939, the red and striped seeds and those from the two branch heads were planted separately. No plants were obtained from one branch head and only a few from the other. The following results were obtained:

Seed-source	*Total plants matured*	*White-seeded plants*
Striped seed from chimera head	66	12
Red seed from chimera head	88	17
Seed from upper branch head (red)	17	4

Each lot of seed produced red-seeded

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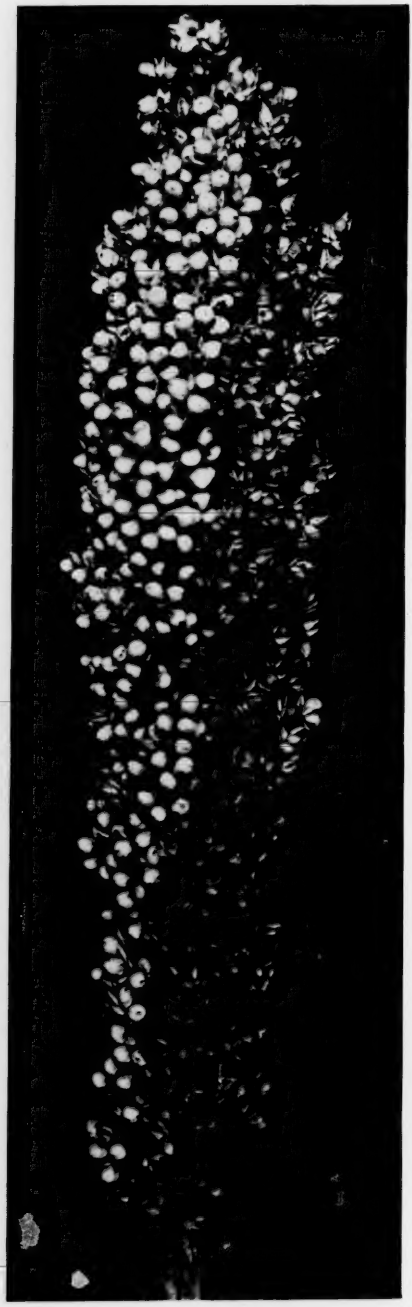
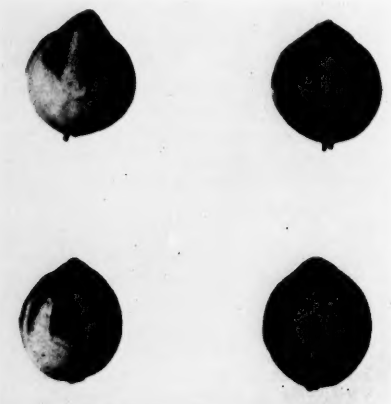


and white-seeded plants in numbers approaching a 3:1 ratio. None of the white seeds produced in 1939 showed the red striping found in the chimera head. It is apparent that the chimeral plant was heterozygous for seed color but that the mutation causing the striped seeds did not affect the genes which determined the seed color in the following generation.

Thus it might be assumed that in the above metroclinal (combination of periclinal and sectorial) chimera, a mutation had occurred in the gene for red color, or in a modifier affecting the expression or distribution of the red in the pericarp, which resulted in striped seeds. Such a mutation must have occurred in a cell which gave rise to pericarp but not to embryo tissue. The probability that the mutation could be expressed without affecting the germ cells is suggested by a case reported by Rhoades* in maize in which the genetic nature of the anther wall was not always identical with that of the enclosed sporogenous tissue.

*Rhoades, M. M. Effect on the *Dt* gene on the mutability of the a_1 allele in maize. *Genetics* 23:377-395. 1938.



TWO KINDS OF SEEDS

Figure 14

On the left is shown the chimeral panicle, nearly natural size. Above are shown the seeds found on this panicle (enlarged), the striped seeds on the left—red on the right.

RECESSIVE POLYDACTYLISM

Associated With Mental Deficiency

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MOST of the cases of human polydactylism have been classified as dominant hereditary traits, with occasionally an irregularity in the expression of the trait due to some unknown circumstances. In an analysis of several histories of polydactylism, Koehler[2] found a close association between the probable type of heredity involved and the expression, symmetrical or asymmetrical, of the defect. The histories which might require an interpretation of incompletely dominant or recessive heredity had a greater tendency for asymmetrical expression than the cases which were clearly dominant. In a few histories polydactylism has been explained as due to recessive factors. This interpretation has been given by Rudert[4] because of the appearance of polydactyl sibs whose family history showed no such anomaly, and also because of the frequent occurrence of polydactyl individuals whose parents were normal, but whose family history showed a relatively high frequency of the anomaly.[5].

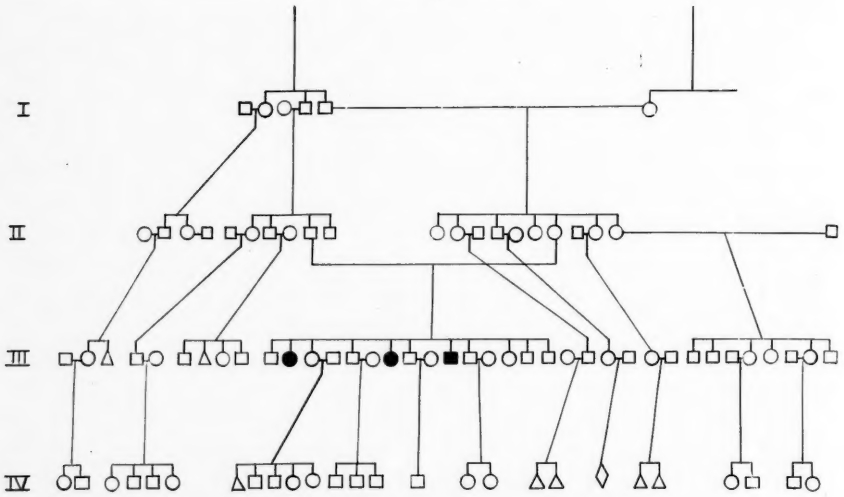
In the history of the family reported here, polydactylism occurs in a family of Scandinavian ancestry.[3] Most of the individuals who are reported in the pedigree (Figure 15) now live in our Northwestern states. The trait occurs in one family of generation III. In this family of eleven children, the second, fifth, and seventh members are defective. Their respective ages in 1939 were fifty, forty-four, and thirty-five. Two of the abnormal individuals are females; and one is a male.

Associated Defects

The three defective persons have similar physical and mental anomalies. Each had an extra digit joined to each hand and foot at the base of the fifth digit. The extra digits have been removed, but the scars remain. There are no extra metacarpal or metatarsal bones. Also the polydactylous members are feebleminded. The oldest of the three has been confined to an institution most of her life. Her defective brother has about the same grade of mental ability, and has been in an institution although he now remains at home. The other girl has learned to write and to read simple words. The three defective persons are slight in body-build, lacking the physical strength which is observed in other members of their family.

One of the defective members, the oldest, was given a physical examination in 1939. Besides the anomalies which have been described, the person has a definite speech defect. Her speech is indistinct, faltering, and scanty in expression. She has also a supernumerary nipple on the left breast. In every other way, the results of the physical examination and of the blood, serological, and urine analyses were those to be expected if the person were classified as a normal individual.

No other member of the family shows any sign of the anomalies. The parents of the children also are normal; and as shown by the family history, the parents are cousins. Relatives of the family also seem to be free of the anomalies. In so far as it can be determined accurately, no condition similar to the mental or physical defects has occurred in the sixty blood-relatives. Members of the clan who have lived in Europe can not recall that any related defects are known from older family records.



ASSOCIATED MENTAL AND PHYSICAL DEFECTS

Figure 15

Pedigree chart showing recessive inheritance of a defect expressed by polydactylism. mental deficiency and physical weakness. The trait appears only in three sibs of the family. Parents of the children are cousins.

Discussion and Conclusions

All the evidence points to the conclusions that in this family the polydactylism and the mental defect developed as a result of heredity, and that a recessive hereditary factor was the basis for the anomalies. Three members of one family have the defects. They did not occur at any definite age of the parents; and normal children occurred between the births of the defective ones. In the three abnormal children, the defects are very similar in nature, varying only in the grade of mentality for one child. As is to be expected if the trait is recessive, the parents of the defective children are normal. It is very significant that the grandfathers of the children were brothers, as shown in generation I. The consanguineous marriage supports an interpretation of recessive Mendelian heredity. A marriage between relatives is likely to bring together in their offspring a rare, recessive, hereditary combination which the parents received from a common ancestor. An interpretation of recessive inheritance is also supported by the absence of similar anomalies in the other members shown in the four generations of the family history. Although the association of the anomalies might be explained as due to linkage, there is no basis for such an interpretation. It would seem to be more logical to interpret the defects as due to a hereditary abnormality of development which was recessive and generalized in nature. The defective developmental process was expressed in three ways: polydactylism, mental deficiency, and a weakened physical condition of the body.

The history in this family differs from the other cases of recessive polydactylism which have been reported. The cases reported by Snyder involved negro families. In the report by Koehler, polydactyl individuals whose histories required an interpretation of recessive heredity tended to show asymmetrical expression of the trait. The recessive history reported by Rudert involved other skeletal defects, including the skull, as well as polydactyly. In the history reported here, the family is Scandinavian; the

polydactylous condition is symmetrical in expression, involving both hands and feet alike; and no other skeletal defect is known. Moreover, it does not seem that the defective individuals in this family belong to the type known as oxycephaly or tower-skull. Although the individuals have the mental involvement and the polydactylism which are usually observed with tower-skull, they lack any trace of the defective skull, obesity, syndactyly, or defective vision, and apparently show no evidence of hypogenitalism.

I express my appreciation to Dr. Magnus C. Peterson, who examined one of the individuals.

Summary

1. In one family, three of eleven children are polydactyl, mentally deficient, and weak physically. The defects are very similar in the three individuals.

2. All other members of the family and all the blood relatives are normal with respect to the three defects.

3. Parents of the defective children are first cousins.

4. The interpretation is made that the anomalies are associated and are caused by a recessive factor.

5. Differences between this case and histories of recessive polydactyly are given.

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HEREDITY AND THE MALFORMED CHILD

IN 1936 Madge Macklin published (*Amer. Jour. Obs. and Gynecol.* 32: 258-265) an analysis of 1420 reports of congenital malformation, from which she selected 72 cases involving twins. In 40 cases only one twin was malformed; both twins were malformed in 32 cases. The sex distribution showed malformation in one twin predominantly among two-egg twins, which strongly suggests that hereditary influences are important in the production of that heterogeneous collection of obscure and diverse medical entities that we customarily lump together as Congenital Malformations.

Of Macklin's 1,420 cases, 1,125 were in families having more than one child, and among these, 311 or 27.6 per cent had two children malformed. In 80 per cent of cases with two children malformed the malformations were identical, again proclaiming that heredity is at the basis of much infant malformation.

An excellent little book has just appeared* which strengthens still further such genetic hunches. It discusses the problem of Congenital Malformations under the headings of Frequency of Malformations, Non-reproductive Characteristics of the Parents, Details of the Defective Child Pregnancy, Characteristics of the Offspring and Etiology of Congenital Malformations.

By looking up death certificates of children dying during the years 1929-1933 the cases of congenital malformation were located and the chief diagnosis selected. Home visiting was done by four fourth year medical students. Because about 80 per cent of these children were born or died in hospitals, these additional records could be consulted as

*Murphy, D. P., Congenital Malformations. Pp. 98. Cloth bound, offset, $2.00. Univ. of Penna. Press, Philadelphia, 1940.

soon as information was received from relatives. The physicians attending the cases were contacted occasionally. This labor yielded 1,476 cases of congenital malformation running 315, 318, 306, 306 and 231 for the separate years.

Among 7,478 stillborn infants were found 222 cases or 297 per 10,000, and among 122,654 live born 668 or 54 per 10,000.

When a pair have had a defective child the chances that they will produce another are 24 times greater than in the general population.

That heredity is involved is further suggested in that two wives each bore to the same man a child having an intestinal defect. A first wife bore to another man a hydrocephalic child and a second bore him one with meningo-myelocele. In 6.9 per cent of the cases Dr. Murphy discovered the existence of such defects in the family of one of the two parents.

Colored persons will rejoice that the incidence of congenital malformations in their group is only half that found in whites, which fact is incidentally a blow to the theory that syphilis causes such malformation, because syphilis is much more common among negroes than among whites. Aryan superiority receives a gentle snub in Dr. Murphy's Figure 1 which shows the country of maternal origin with the England-Scotland-Wales group having the greatest percentage of malformations followed by the Denmark-Norway-Sweden group; then Germany with Ireland as a close runner-up.

Among the destitute the rate is more than twice that founud in the general population. Only 1.8 per cent reported consanguinity—of all degrees.

When mothers were divided into two groups with average ages of 16.8 years and 26.8 years respectively then the mother of the first group has her first child about 15 months later and her defective child at about 90 months, whereas the woman of the older group has her first child at about 21 months and her defective child at 59 months. There is an increase in ratio of defective children to normal sibs with increase in size of family. This difference suggests a possible intrauterine environmental role, the percentage of defective children to normal siblings increasing obviously with the age of the mother.

Some of the detailed classifications of malformation employed were:

Hydrocephalus alone	Craniorachischisis
Spina bifida alone	Microcephalus
Anencephalus	Encephalocele
Hydrocephalus	Mongolism.
Meningocele	

Of these, the first four make up 85.7 per cent of the nervous anomalies.

Of the alimentary tract, atresia and obstruction form the chief malformations Piloric stenosis alone composing 46.9 per cent of the total and atresia of the esophagus small intestine and anus making up 24.7 per cent more.

Of the Cutaneo-muscule-skeletal system, harelip and cleft palate make up 44.7 per cent of the malformations, followed by gastroschisis (13.6%), maldevelopment of extremities (11.7%), Hernias (11.6%), Hernia or absence of diaphragm (6.7%), dislocation of the hips (3.6%).

Defects of the genito-urinary system were found in ten cases, defects of the respiratory system in three, and ill defined defects in four.

When malformation was found among relatives the relationship of malformed relative to the propositus was usually cousin, uncle or aunt. The viability of malformed children is poor, 90.6 per cent dying during the first year.

In the chapter on Etiology Dr. Murphy states cautiously, "It is said that development results from the interaction between inherited tendencies contained within the egg substance itself, and the external conditions which surround and act upon this substance. It is common knowledge that many congenital abnormalities are inherited."

No evidence of environmental factors was found.

This splendid little book will be of value to all persons seeking to advance the rapidly developing subject of Medicogenetics.

Clyde E. Keeler.

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Slide of Normal Salivary Chromosomes of *D. melanogaster*, each	$2.25
Three Slides	5.00
Slide with drawing identifying all chromosomes in two figures (Female)	4.00*
Slide showing Y-chromosome (Male)	4.00
Slide showing synapsis of normal and inverted X-chromosome (loop)	4.00*
Slide showing figure of an autosomal inversion	4.00*
Slide showing synapsis of translocated and normal chromosomes	4.00*
Slide showing a small deficiency (deletion)	4.00*
Salivary chromosomes of *D. virilis*	2.25
Three Slides	5.00
Normal Somatic (ganglion) chromosomes of *D. melanogaster* (*Magnification of at least 1000 × necessary to view satisfactorily.*)	2.50
1 Slide of each of above (9 slides—6 drawings)	25.00

* Three or more slides, 1 of each kind @ $3.50 each. Three or more of same kind @ $3.00 each.

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Painter's cytogenetic map of the salivary chromosomes, 9½ by 18 inches, line-cut showing major chromosome details and the genes approximately located to end of 1934, mailed unfolded	$ .50
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Dobzhansky's map of Salivary Chromosomes of *Drosophila Azteca* (9½ by 18)	.75
1 copy each of Painter's Hughes' Dobzhansky's, and Bridges' two maps (5 maps in all)	3.00

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† Volume and page number of illustration in the JOURNAL OF HEREDITY.

ILLUSTRATED LITERATURE

"Symposium Reprint" on Salivary Gland Chromosomes, containing articles from the JOURNAL OF HEREDITY, 1934-1938, by Painter, Bridges, Metz, Mackensen and Hughes—four Salivary Gland Chromosome maps, 15 illustrations, 54 pages $3.00

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